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## Percutaneous Transluminal Renal Angioplasty (PTRA) and Surgical Revascularisation in Renovascular Disease—A Retrospective Comparison of Results, Complications, and Mortality

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**Objective.** To evaluate results, complications and mortality following percutaneous transluminal renal angioplasty (PTRA) and open surgical revascularisation for renovascular disease.

**Methods.** A retrospective evaluation of 381 renovascular patients (median age 64, range 9–99 years, 152 women) treated at Malmö University Hospital during 1987–1996. Two hundred and sixty-two (69%) of the patients were treated with PTRA, 106 (28%) with open revascularisation.

**Results.** Thirty-day mortality was 2% in the PTRA group and 9% after open surgery ( $p < 0.001$ ). There were no differences between groups concerning the number of re-do procedures, but first re-do was performed after seven (IQR 3–14) months in the PTRA group, and after 15 (IQR 10–44) months after open revascularisation ( $p < 0.0001$ ). After a median follow-up of 4 months (IQR 0–13) systolic and diastolic blood pressure (BP) had decreased ( $p < 0.0001$ ) in both groups. The number of antihypertensive drugs was reduced ( $p < 0.0001$ ) and S-creatinine levels were unchanged in both groups. Long-time survival assessed with log-rank analysis was better ( $p < 0.01$ ) in the PTRA group. The risk ratio for death with open revascularisation was 1.69 ( $p < 0.01$ ).

**Conclusions.** In this retrospective comparison, PTRA was as effective as open revascularisation, with lower complication rate and lower early and long-time mortality, but with shorter time to first re-do.

**Key Words:** Renal artery stenosis; PTRA; Open renal vascularisation; Long-term follow-up.

### Introduction

Renal artery stenosis is known to cause hypertension or renal insufficiency, or both. Natural history studies have shown progressive atherosclerotic disease in 44–49% of individuals with atherosclerotic renal artery stenosis, even a little higher if associated with aortic atherosclerotic disease. Occlusion occurs in 7–16% during a 5–7 year follow-up, the risk of occlusion being particularly great in stenosis  $> 75\%$ .<sup>1,2</sup> In fibromuscular dysplasia stenosis is progressive in 34%.<sup>1</sup>

Indications for revascularisation are to control hypertension, improve renal function, or prevent renal failure, and occasionally ‘flash’ pulmonary oedema, especially in single renal patients. The treatment of choice in such cases was traditionally

and remains so in certain centres—open surgery,<sup>3–11</sup> but during the last decade, percutaneous transluminal renal angioplasty (PTRA) has emerged as an alternative treatment.<sup>10,12–17</sup>

Weibull *et al.*, proposed the following conditions for accepting PTRA as a primary method for revascularisation.<sup>10</sup> ‘Technical success  $> 80\%$ , two-year primary patency  $> 75\%$ , two-year secondary patency the same as for surgery, and the same results on blood pressure and renal function as for open surgery’.

As these conditions were fulfilled, PTRA was recommended as first choice of treatment at our institution.<sup>10</sup> The intention of the present study was to follow up the clinical practice, which, with growing experience, has emerged from this recommendation, and evaluate the available methods for treatment of renal artery stenosis, i.e. open revascularisation and PTRA, in a long-term perspective. Complications, mortality, and short- and long-term results for the different methods are described and compared for all

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patients treated at Malmö University Hospital in 1987–96.

## Material and Methods

### Methods

All 381 patients that underwent surgical or endovascular procedures for renovascular disease at Malmö University Hospital during 1987–1996 were identified and patient files from departments involved in the management were reviewed. Complementary information from the Swedish Vascular Registry (Swed-vasc) was obtained for 297 (78%) patients and from a patient questionnaire (in average 7.1 years after treatment) in 145 patients. Regarding BP and s-creatinine only data from hospital records were utilised. Data on survival for all patients were obtained in 2001.

### Patient material

Of the 381 patients (median age 64, range 9–99 years, 152 women) 262 (69%) were primarily treated with PTRa, 106 (28%) with open revascularisation, of which 33 (31%) had simultaneous measures against aneurysm or occlusive disease of the abdominal aorta. Open revascularisation was normally a transverse arteriotomy, endarterectomy and patch closure if not aortic surgery was needed when a 5–6 mm dacron or PTFE by-pass with end-to-end anastomosis to the renal artery was used. The remainder underwent nephrectomy ( $n = 11$ ), division on the crus diaphragma ( $n = 1$ ) and correction of a venous malformation ( $n = 1$ ). Of the 262 patients undergoing PTRa, 24 patients during the last two years studied had stent placement (Table 1).

Open revascularisation dominated during 1987–90, accounting for 60% of primary treatments, whereas PTRa accounted for > 90% during 1993–96. The cause of stenosis was atherosclerosis in 328 (86%) patients (median age 65 years, 118 women) and fibromuscular dysplasia (FMD) in 40 (10%) patients

(median age 40 years, 31 (78%) treated with PTRa, 34 women, Table 1). Five patients (three treated with PTRa) had renal transplants. Other diagnoses were venous and arterial malformation, radiation injury, external compression of the renal artery by crus diaphragma and Tachayasu's disease. Bilateral treatment was performed in 138 (36%) patients (Table 1).

The main indication for treatment were hypertension and renal function only in 17 (6%) in the PTRa-group and 10 (9%) in the open revascularized group. Clearance was not regularly determined. Risk factors such as smoking, diabetes, hyperlipidemia, and occurrence of earlier vascular operations, amputations, and cardiovascular or cerebrovascular disease were insufficiently recorded, but when reported, there were no differences between the groups.

Renal diseases, such as chronic glomerulonephritis, diabetic nephropathy, polycystic kidneys etc., occurred in seven (3%) patients treated with PTRa and in two (2%) openly treated patients, nephrosclerosis in one patient each.

All secondary procedures aiming at improving renal artery flow after the initial treatment, also including the contralateral renal artery, was considered as a re-do procedure.

### Definitions of outcome

Effects on blood pressure were regarded as:<sup>18</sup>

Cure—diastolic BP < 90 mmHg and systolic BP < 140 mmHg, off antihypertensive medication.

Improvement—diastolic BP < 90 mmHg and/or systolic BP < 140 mmHg on the same or reduced number of medications or a reduction in diastolic BP by at least > 15 mmHg with the same or a reduced number of medications.

Failure—no change or inability to meet the criteria for cure or improvement.

Renal function was regarded as:

Improvement—s-creatinine was reduced by  $\geq 20\%$ .

Stabilisation—s-creatinine changed by < 20%.

Failure—s-creatinine increased by  $\leq 20\%$ .

**Table 1.** Patient material,  $n$  (%) or median and range.

	PTRa ( $n = 262$ )	Open revascularisation ( $n = 106$ )	Nephrectomy ( $n = 11$ )	Other operation ( $n = 2$ )
Sex (M/F)	148/114	69/37	7/4	2/0
Age (years)	65 (9–99)	64 (9–84)	62 (52–73)	19 (17–21)
Atherosclerotic stenosis	231 (88)	86 (81)	11 (100)	0 (0)
Fibromuscular dysplasia	31 (12)	9 (8)	0 (0)	0 (0)
Bilateral intervention	99 (38)	39 (37)	0 (0)	0 (0)

*Statistical analysis*

Results are reported as median and interquartile range. Comparisons between patients undergoing PTRA and reconstructive surgery were made with the Mann–Whitney U-test or the Chi<sup>2</sup> test, and comparisons within groups with the Wilcoxon's signed rank test. Long-time survival curve was estimated according to Kaplan–Meier, and the comparison between groups was made with log-rank analysis. Risk ratio for death was evaluated with Cox proportional regression analysis. Because of the limited number of patients undergoing nephrectomy and other operations, no statistical calculations were made in these two groups. *P* values < 0.05 were considered significant. Stat View 4.5 (Abacus Concepts Inc., Berkeley, CA, USA) was used for the statistical calculations.

**Results***Early mortality and complications*

Thirty-day mortality was five (2%) in the PTRA group and 10 (9%) after open surgery (*p* < 0.0001). Reconstructive complications were recorded or denied for all patients during hospital stay but 30 day follow-up data was obtained for 266 (70%) patients, 164 (63%) in the PTRA group and 92 (87%) openly reconstructed (Table 2). Infection was more common among patients undergoing open reconstruction (*p* < 0.05). There were no differences between groups concerning the number of general complications (Table 3). The overall procedural complication rate was 9% in the PTRA group and 22% among openly reconstructed patients (*p* < 0.05). Within the open group, the complication rate was significantly (*p* < 0.05) higher when combined renal and aortic reconstruction was done, 11/33 (35%) compared with 12/73 (15%) when only renal artery reconstruction was made.

Early adverse results (deterioration or death within a month) occurred in 28 patients treated with PTRA (10%), 14 treated with open renal artery surgery (19%) and five patients treated with aortic and renal surgery (15%).

*Blood pressure and renal function*

Data on BP, s-creatinine and the number of antihypertensive drugs could be obtained from patient files for up to median four (IQR 0–14) months. Both systolic and diastolic BP decreased (*p* < 0.0001) in both patients treated with PTRA (from 185 (168–200)/100 (90–110) to 150 (140–170)/85 (80–90) mmHg) and open revascularisation (from 180 (160–202)/100 (90–110) to 150 (140–170)/90 (80–90) mmHg). Moreover, the number of antihypertensive drugs was significantly (*p* < 0.0001) reduced from 2 (2–3) to 2 (1–3) in the PTRA group, and from 2 (2–3) to 1 (1–2) in the openly revascularised group. The s-creatinine levels, on the other hand, were unchanged, 123 (92–172) and 115 (90–172) µmol in the PTRA group, and 125 (100–180) and 125 (98–197) µmol in the openly revascularised group.

When the results were categorized as above, benefit (cure or improvement) regarding hypertension was achieved in 172 (66%) patients treated with PTRA after an average of 1.4 procedures per patient, and in 64 (60%) patients undergoing open revascularisation after 1.3 procedures per patient. Of those categorised as failure regarding control of hypertension 41 out of 90 PTRA-patients and 26 out of 52 open revascularised patients had there antihypertensive treatment reduced. Renal function was improved or stabilised for 217 PTRA-patients (83%) and failure was seen in 45 (17%). For open revascularisation renal function was improved or stabilised for 68 patients (64%) and failure noted for 38 patients (36%) (*p* < 0.001).

Late deterioration was most common after open revascularisation without aortic surgery (11%, 2.25 procedures per patient) to be compared with PTRA

**Table 2. Re-dos and reconstructive complications (median and IQR or *n* (%)).**

	PTRA ( <i>n</i> = 262)	Open revascularisation ( <i>n</i> = 106)
Number of re-do procedures	108	30*
Number of patients needing re-do (%)	75 (29)	21 (20)
Number of patients needing > 1 re-do	34 (13)	9 (8)
Time to 1: st re-do (months)	7 (3–14)	15 (10–44)**
Reconstructive complications	PTRA (30 day reported for 164)	Open revascularisation (30 day reported for 92)
Bleeding/haematoma	4 (2)	7 (8)
Occlusion/thrombosis	7 (4)	6 (6)
Infection	0 (0)	3 (3)*
Distal embolisation	2 (1)	2 (2)

\**p* < 0.05 compared with the PTRA group, \*\**p* < 0.0001 compared with the PTRA group.

**Table 3. General complications and mortality (n (%)).**

General complications	PTRA (n = 262) (30 day reported for 143)	Open revascularisation (n = 106) (30 day reported for 96)
Renal complications	15 (9)	12 (12)
Cerebrovascular complications	3 (2)	1 (1)
Cardiac complications	3 (2)	4 (4)
Multi-organ failure	0 (0)	2 (2)
Pulmonary complications	1 (1)	2 (2)
Sepsis	0 (0)	1 (1)
Intensive care > 5 days	1 (1)	4 (4)
Mortality	PTRA (n = 262)	Open revascularisation (n = 106)
30-day mortality	5 (2)	10 (9)**

\* $p < 0.05$  compared with the PTRA group, \*\* $p < 0.001$  compared with the PTRA group.

(8%) and open revascularisation with aortic surgery (6%), both groups at 1.5 procedures per patient.

#### *Re-do procedures (Table 2)*

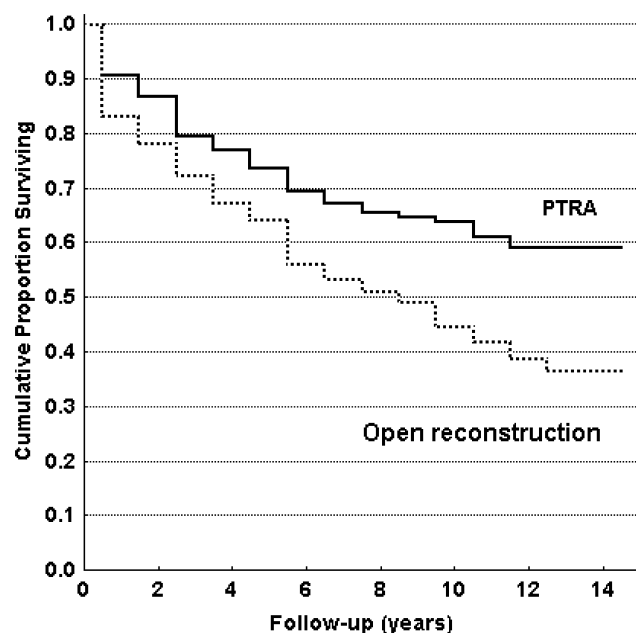
The number of re-do procedures, but not the number of patients undergoing re-do, was significantly higher in the PTRA group ( $p < 0.05$ , Table 2). Furthermore, the first re-do was performed after seven (3–14) months in the PTRA group, compared with 15 (10–44) months after open revascularisation ( $p < 0.0001$ ). Of the 75 PTRA-patients requiring a re-do 59 had re-do PTRA (79%), and of the 21 open revascularised patients requiring re-do 14 had re-do PTRA (67%). Those requiring more than one re-do procedure had repeated re-do with PTRA in 32/33 PTRA-patients (92%) versus 7/9 open revascularised patients (75%).

#### *Long term mortality (Fig. 1)*

Long-time survival was significantly ( $p < 0.01$ ) better in the PTRA group (Fig. 1), with a risk ratio for death of 1.69 ( $p < 0.01$ ) in the openly reconstructed group.

The number of patients under observation were:

Number of observation years	Number of observed PTRA patients	Number of observed open revascularisation patients
0	262	106
2	226	84
4	200	73
6	142	62
8	81	54
10	42	40
12	22	23



**Fig. 1.** Kaplan–Meier long-time survival curve for patients after PTRA ( $n = 262$ , solid line) and after open reconstruction ( $n = 106$ , dotted line). Yearly assessments of survival.  $P < 0.01$  for difference between groups.

## **Discussion**

In our analysis PTRA appears to be as effective as open surgery for the treatment of isolated renal artery stenosis. In addition, mortality for PTRA-treated patients in the present population was reduced both at 30 days and in long-term follow-up. Moreover, the risk of deterioration was significantly less for PTRA-treated, and the difference in the number of re-do procedures—required to achieve the desired result—was not different as presumed. A major flaw and source for bias in this study is that the majority open surgical patients were treated in the 80s and the majority of PTRA-patients in the 90s. A simple analysis of risk factors and patient's characteristics does not reveal any major differences between the groups.

Procedural mortality was 2% for PTRA and 9% for open revascularisation ( $p > 0.01$ ). A number of other studies support this difference. In large studies open



revascularisation has shown a 30-day mortality of 5.5–7.3%, also including a large proportion of patients undergoing subsequent aortic surgery as well.<sup>3,5</sup> This has to be compared with the reported 30-day postprocedural PTR mortality of 0–1.5%,<sup>15,18,20</sup> corresponding well with our results. Thus, PTR has lower procedural morbidity/mortality. With today's refined endovascular techniques, if combined aortic and renal disease is seen, optimisation of the renal arteries before aortic reconstruction seems preferable. Even in patients with aneurysm, we would advocate to have stenosis of the renal arteries endovascularly dilated, and if required, stented prior to surgery. At least theoretically, an optimised renal function should decrease the operative risk.

In the PTR group the long-time survival was 74% at five years and 64% at 10 years corresponding to a five-year survival of 64% and 10-year of 45% in the open revascularised group. Survival in other large surgically treated groups with equal age distribution has been reported to be 60% at 38 months<sup>3</sup> and 71% at 5 years.<sup>5</sup> In other studies of PTR a four-year survival of 74% was reported,<sup>21</sup> however, Paulsen and co-workers found a five-year survival of only 58%.<sup>15</sup> Thus, our finding is difficult to explain and not entirely supported by other reports. It is, though, very difficult to compare figures from different institutions. The same selection criteria for treatment were used during the analysed period in our institution, indicating that our results might well be an important finding.

Re-do procedures were done earlier after PTR than after open surgery, which may partly be due to re-dos being more readily expected and accepted after PTR. Moreover, many of our patients were followed with routine control angiography,<sup>14</sup> which in some cases may have led to re-intervention without clinical signs of re-stenosis. Additionally, patients with bilateral disease often had their endovascular treatment staged in two procedures adding to a higher incidence of re-do procedures in the PTR group as well, since also procedures to the contralateral kidney were considered as re-dos. Further improved results using stents especially for ostial lesions have been reported.<sup>19–22</sup>

Re-dos were concentrated to a fraction of the population. In order to choose the proper first line treatment, individualise follow-up and prevent recurrences, it would be valuable to further characterise those more likely to need a re-do procedure. Originally, routine control angiography was done after 3–6 months. But with growing experience a less rigorous practice has emerged, and our study comprises mainly patients managed according to this practice. We believe that patients with a remaining pressure

gradient after a PTR have a three to four times increased risk of developing re-stenosis and only this group, nowadays, routinely undergoes control angiography.

Hypertension can often be successfully managed with modern antihypertensive drugs and, therefore, surgical or endovascular intervention has been questioned.<sup>23</sup> However, as secondary hypertension, including renovascular hypertension often features a blunted nocturnal fall in blood pressure,<sup>24</sup> it is important to reduce nocturnal blood pressure, to avoid increased workload for the heart causing increased long-term cardiovascular morbidity/mortality. Moreover, lowering the blood pressure with effective medical therapy in a patient with a significant stenosis might reduce renal blood-flow, leading to hypoperfusion and risk of ischemic atrophy or even infarction. The main problem, therefore, seems to be to evaluate when a renal artery stenosis is of haemodynamic importance and should be treated more aggressively. The currently ongoing studies ASTRAL and STAR will be important to elucidate which renal artery stenosis should be selected for treatment, but long-term follow-up seems to be needed for the evaluation of morbidity/mortality and what type of treatment that will be optimal.

Clinical criteria are not sufficient to select patients for renal artery treatment. Renogram and duplex scanning are not sensitive enough. MRA and CT angiography are under rapid improvement, and will probably be beneficial screening tools for tomorrow. Evaluation of the degree of stenosis from angiography may be insufficient. During the last 10-year period, we have partially based our judgement on treatment or not on pressure recordings during angiography, comparing the pressure in the aorta and in the renal arteries, and have found this to be as valuable as reported by other groups.<sup>25</sup> We are currently studying whether pressure gradients will be useful in selecting those who need treatment.

Only few studies have randomised patients to different treatments.<sup>10,23</sup> Randomised studies with strict inclusion criteria are needed for both short- and especially also long-term comparisons of endovascular treatment with 'only' medical treatment for 'success', morbidity and mortality. In this analysis of a single-centre experience, it seems as endovascular treatment compared with open revascularisation gives at least equal results concerning hypertension and renal function, but the finding of a reduced procedural and long-term morbidity/mortality for endovascular treatment makes it the first line of treatment.

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### References

- ERDOES LS, BERMAN SS, HUNTER GC, MILLS JL. Comparative analysis of percutaneous transluminal angioplasty and operation for renal revascularization. *Am J Kidney Dis* 1996; **27**: 496–503.
- CAPS MT, PERISSINOTTO C, ZIERLER RE, POLISSAR NL, BERGELIN RO, TULLIS MJ, CANTWELL-GAB K, DAVIDSON RC, STRANDNESS JR. DE. Prospective study of atherosclerotic disease progression in the renal artery. *Circulation* 1998; **98**: 2866–2872.
- HANSEN KJ, CHERR GS, CRAVEN TE, MOTEW SJ, TRAVIS JA, WONG JM, LEVY PJ, FREEDMAN BI, LIGUSH JR. J, DEAN RH. Management of ischemic nephropathy: dialysis-free survival after surgical repair. *J Vasc Surg* 2000; **32**: 472–482.
- KUESTNER LM, STONEY RJ. The case for renal revascularization. *Cardiovasc Surg* 1995; **3**: 141–154.
- DARLING III RC, KREINENBERG PB, CHANG BB, PATY PS, LLOYD WE, LEATHER RP, SHAH DM. Outcome of renal artery reconstruction. Analysis of 687 procedures. *Ann Surg* 1999; **230**: 524–532.
- DERROW AE, SEEGER JM, DAME DA, CARTER RL, OZAKI CK, FLYNN TC, HUBER TS. The outcome in United States after thoracoabdominal aortic aneurysm repair, renal artery bypass, and mesenteric revascularization. *J Vasc Surg* 2001; **34**: 54–61.
- PATY PS, DARLING III RC, LEE D, CHANG BB, RODDY SP, KREINENBERG PB, LLOYD WE, SHAH DM. Is prosthetic renal artery reconstruction a durable procedure? An analysis of 498 bypass grafts. *J Vasc Surg* 2001; **34**: 127–132.
- REIHER L, PFEIFFER T, SANDMAN W. Long-term results after surgical reconstruction for renal artery fibromuscular dysplasia. *Eur J Vasc Endovasc Surg* 2000; **20**: 556–559.
- CHERR GS, HANSEN KJ, CRAVEN TE, EDWARDS MS, LIGUSH JR. J, LEVY PJ, FREEDMAN BI, DEAN RH. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg* 2002; **35**: 236–245.
- WEIBULL H, BERGQVIST D, BERGENTZ S-E, JONSSON K, HULTÉN L, MANNHEM P. Percutaneous transluminal renal angioplasty vs. surgical reconstruction of atherosclerotic renal artery stenosis—a prospective randomised study. *J Vasc Surg* 1993; **18**: 841–852.
- VAN ROODEN CJ, VAN BOECKEL JH, DE BACKER GG, HERMANS J, CHANG PC. Long-term outcome of surgical revascularization in ischemic nephropathy: Normalization of average decline in renal function. *J Vasc Surg* 1999; **29**: 1037–1049.
- SOS TA, PICKERING TG, SNIDERMAN K, SADDEKNI S, CASE DB, SILANE MF, VAUGHAN JR. ED, LARAGH JH. Percutaneous transluminal renal angioplasty in renovascular hypertension due to atheroma or fibromuscular dysplasia. *N Engl J Med* 1983; **309**: 274–279.
- WEIBULL H, BERGQVIST D, JONSSON K, CARLSSON S, TAKOLANDER R. Analysis of complications after transluminal angioplasty of renal artery stenosis. *Eur J Vasc Surg* 1987; **1**: 77–84.
- WEIBULL H, BERGQVIST D, JONSSON K, HULTÉN L, MANNHEM P, BERGENTZ S-E. Long-term results after percutaneous transluminal angioplasty of atherosclerotic renal artery stenosis—the importance of intensive follow-up. *Eur J Vasc Surg* 1991; **5**: 291–301.
- PAULSEN D, KLOW NE, ROGSTAD B, LEIVESTAD T, LIEN B, VATNE K, FAUCHALD P. Preservation of renal function by percutaneous transluminal angioplasty in ischaemic renal disease. *Nephrol Dial Transplant* 1999; **14**: 1454–1461.
- BUSH RL, NAJIBI S, MACDONALD MJ, LIN PH, CHAIKOF EL, MARTIN LG, LUMSDEN AB. Endovascular revascularization of renal artery stenosis: technical and clinical results. *J Vasc Surg* 2001; **33**: 1041–1049.
- GEROULAKOS G, MISSOURIS C, MITCHELL A, GREENHALGH RM. Endovascular treatment of renal artery stenosis. *J Endovasc Ther* 2001; **8**: 177–185.
- RUNDBACK JH, SACKS D, KENT KC, COOPER C, JONES D, MURPHY T, ROSENFELD K, WHITE C, BETTMANN M, CORTELL S, PUSCHETT J, CLAIR DG, COLE P, FOR THE AMERICAN HEART ASSOCIATION. Guidelines for the reporting of renal artery revascularization in clinical trials. *J Vasc Intervent Radiol* 2002; **13**: 959–974.
- DORROS G, JAFF M, MATHIAK L, HE T. Multicenter Palmaz stent renal artery stenosis revascularization registry report: four year follow-up of 1058 successful patients. *Cath Cardiovasc Int* 2002; **55**: 182–188.
- LEDERMAN RJ, MENDELSON FO, SANTOS R, PHILLIPS HR, STACK RS, CROWLEY JJ. Primary renal stenting: characteristics and outcome after 363 procedures. *Am Heart J* 2001; **142**: 314–323.
- BAUMGARTNER I, VON AESCH K, DO DD, TRILLER J, BIRNER M, MAHLER F. Stent placement in ostial and nonostial atherosclerotic renal artery stenosis: a prospective follow-up study. *Radiology* 2000; **216**: 498–505.
- BAKKER J, GOFFETTE PP, HENRY M, MALI WP, MELKI JP, MOSS JG, RABBIA C, THERASSE E, THOMSON KR, THUMBER S, VIGNALI C. The ERASME study: a multicenter study on the safety and technical results of the Palmaz stent used for the treatment of atherosclerotic ostial renal artery stenosis. *Cardiovasc Intervent Radiol* 1999; **22**: 468–474.
- VAN JAARSVELD BC, KRIJNEN P, PIETERMAN H, DERKX FH, DEINUM J, POSTMA CT, DEES A, WOITTEZ AJ, BARTELINK AK, MAN IN'T VELD AJ, SCHALEKAMP MA. The effect of balloon angioplasty on hypertension in atherosclerotic renal-artery stenosis. Dutch renal artery stenosis intervention cooperative study group. *N Engl J Med* 2000; **342**: 1007–1014.
- POLONIA J, SANTOS AR, GAMA GM, BARROS H. Accuracy of twenty-four-hour ambulatory blood pressure monitoring (night-day values) for the diagnosis of secondary hypertension. *J Hypertens* 1995; **13**: 1738–1741.
- NAHMAN JR. NS, MANIAM P, HERNANDEZ JR. RA, FALKENHAIN M, HEBERT LA, KANTOR BS, STOCKUM AE, VANAMAN ME, SPGOS DG. Renal artery pressure gradients in patients with angiographic evidence of atherosclerotic renal artery stenosis. *Am J Kid Dis* 1994; **24**: 695–699.

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